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Emergency medicine updates: Cardiac arrest medications

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ABSTRACT

Introduction: Cardiac arrest is a serious condition frequently managed in the emergency department (ED). Medications are a component of cardiac arrest management.
Objective: This paper evaluates key evidence-based updates concerning medications used for patients in cardiac arrest.
Discussion: Several medications have been evaluated for use in cardiac arrest. Routes of administration may include intravenous (IV) and intraosseous (IO). IV administration is recommended, though if an attempt at IV access is unsuccessful, IO access can be utilized. Epinephrine is a core component of guidelines, which recommend 1 mg in those with shockable rhythms if initial CPR and defibrillation are unsuccessful, while in nonshockable rhythms, guidelines recommend that epinephrine 1 mg be administered as soon as feasible. While epinephrine may improve rates of ROSC, it is not associated with improved survival with a favorable neurologic outcome. Evidence suggests the combination of vasopressin, steroids, and epinephrine may improve ROSC among those with in-hospital cardiac arrest, but there is no improvement in survival to discharge and survival with a favorable neurologic outcome. Antiarrhythmics (e.g., amiodarone, lidocaine, procainamide) likely do not improve short-term or long-term survival or neurologic outcomes, though guidelines state that amiodarone may be used in those with cardiac arrest and refractory pulseless ventricular tachycardia (pVT)/ventricular fibrillation (VF). Calcium and sodium bicarbonate should not be routinely administered in those with cardiac arrest. Beta-blockers may be considered in those with shock-resistant pVT/VF.
Conclusions: An understanding of literature updates concerning medication use in cardiac can improve the ED care of these patients.

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1. Introduction

Cardiac arrest is a condition frequently managed in the emergency department (ED) and is due to loss of organized cardiac function and systemic circulation. The annual incidence ranges between 55 and 113 per 100,000 population, and in the United States there are up to 450,000 patients per year who experience out-of-hospital cardiac arrest (OHCA) [1-10] Mortality is severe, with less than 10 % of patients experiencing OHCA treated by emergency medical services (EMS) surviving to hospital discharge [11].

There are several components of management, including cardiopulmonary resuscitation (CPR) with high-quality chest compressions and early defibrillation in shockable rhythms. Several medications are a component of cardiac arrest management and recommended by guidelines. This review is part of a series discussing evidence-based medicine updates concerning the management of cardiac arrest. This current review will discuss medications in cardiac arrest.

2. Discussion

2.1. What is the recommended route of medication administration in cardiac arrest?

There are several routes for medication administration in cardiac arrest, including intravenous (IV), intraosseous (IO), oral, endotracheal, and intramuscular (IM), though the primary routes are IV or IO [1,6,7,12,13]. IO access has been suggested to provide an easier means of vascular access, and several studies suggest higher success rates and faster time to access with IO. A study with 182 OHCA patients found higher success rates with tibial IO access compared to IV access (91 % versus 43 %) and a trend to faster time to initial access with tibial IO access compared to IV (4.6 min versus 7.0 min) [14], while another study with 112 OHCA patients found higher success rates with IO versus IV

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access (100 % versus 33 %, adjusted odds ratio [OR] 32.4, 95 % confidence interval [CI] 1.8–570.9) [15]. However, the 2025 PARAMEDIC-3 trial found the median time from EMS personnel arrival to vascular access was 12 min in both the IO and IV group, and the time to medication administration was similar (14 min versus 15 min) [16]. The 2025 IVIO trial found median time to success for IO and IV access was also similar (14 min), and the time to first dose of epinephrine was 15 min in both groups [17].

Multiple studies have evaluated the IV versus IO route and whether the route of medication administration is associated with return of spontaneous circulation (ROSC) or survival with favorable neurologic outcome. A meta-analysis of 9 retrospective observational studies with 111,746 adult OHCA patients found no difference in survival with favorable neurologic outcome at discharge between the IO and IV route (OR 0.60; 95 % CI 0.27–1.33) [18]. Subgroup analysis revealed time to intervention was positively associated (OR 3.95, 95 % CI 1.42–11.02) with favorable neurologic outcome, with higher rates in patients receiving IV versus IO administration when time-to-intervention was minimized [18]. However, observational data can only suggest correlation and not causation, and there are several factors including selection bias that may have influenced the results. The 2024 VICTOR trial randomized 1771 patients with OHCA to medications administered via IV versus IO. Authors found no difference in patients discharged alive, prehospital ROSC, sustained ROSC, and survival with favorable neurologic outcome [19]. The 2025 PARAMEDIC-3 trial included 6082 patients randomized to IV versus IO access for medication administration [16]. There was no difference in favorable neurologic outcome at discharge (adjusted OR 0.91, 95 % CI 0.57–1.47), though ROSC was higher in the IV versus IO group (39.1 % versus 36.0 %, adjusted OR 0.86, 95 % CI 0.76–0.97) [16]. The 2025 IVIO study analyzed 1479 patients with OHCA. While success rates for obtaining vascular access within two attempts was higher in the IO group (92 % vs. 80 %), there was no difference in sustained ROSC, survival at 30 days, or favorable neurologic outcome at 30 days [17].

Additionally, IO access may be associated with harms such as dislodgement, inadequate flow rate, extravasation, compartment syndrome, infection (e.g., cellulitis, osteomyelitis), and inability to remove a bent needle, though these are uncommon [20-22].

IM administration of epinephrine has recently been evaluated for use in OHCA. A 2021 before-and-after feasibility evaluated first-dose IM epinephrine in 99 patients [23]. Time to medication administration following call receipt was faster for the IM route compared with IV or IO (time savings of 3 min, 95 % CI 2–4 min), with similar rates of survival to hospital discharge [23]. A study published in 2024 evaluated single dose epinephrine 5 mg IM using a before-and-after design (preintervention period 2010–2019, postintervention period 2019–2024) [24]. Among 1405 patients with OHCA, 420 patients (29.9 %) received IM epinephrine, while 985 patients (70.1 %) received usual care. Time to administration was faster for those receiving IM epinephrine (median 4.3 min, interquartile range [IQR] 3.0–6.0 min) versus standard administration (7.8 min, IQR 5.8–10.4 min). Authors state that IM epinephrine was associated with improved survival to hospital admission (37.1 % versus 31.6 %, adjusted OR 1.37, 95 % CI 1.06–1.77), hospital survival (11.0 % versus 7.0 %, adjusted OR 1.73, 95 % CI 1.10–2.71), and favorable neurologic status at hospital discharge (9.8 % versus 6.2 %, adjusted OR 1.72, 95 % CI 1.07–2.76) [24]. However, this was a before-and-after study, with multiple confounders. The study took place over 14 years, and there have been many changes in OHCA management over that period (e.g., airway, temperature management). Patients in the postintervention period had an approximately 14 % higher rate of bystander CPR, and this group was on average 3 years younger. These issues limit the study conclusions.

Based on current evidence, the most feasible means of access should be obtained, depending on factors such as the setting, resources, and available personnel. Current guidelines recommend IV administration as the preferred route, which may provide the most predictable medication response [7,13]. However, if IV access is not feasible or not successful, IO access should be utilized. Further study is needed concerning IM epinephrine in OCHA prior to routine use.

2.2. What is the utility of epinephrine in cardiac arrest?

Epinephrine is a sympathomimetic catecholamine with alpha-1, alpha-2, beta-1, and beta-2 activity. In patients with cardiac activity, epinephrine may improve coronary and cerebral perfusion and oxygen delivery with its alpha-adrenergic activity, which may increase the likelihood of ROSC [25]. The beta-adrenergic activity can increase heart rate and contractility, though this can also increase cardiac oxygen demand and lead to arrhythmia [25-27]. Despite these potential issues, epinephrine is currently the only medication supported by guidelines in all patients with cardiac arrest, with the Advanced Cardiac Life Support (ACLS) guidelines recommending epinephrine 1 mg IV or IO every 3–5 min [6-9]. Doses greater than 1 mg have been evaluated for use in cardiac arrest, but they are not recommended based on the literature. Several randomized controlled trials (RCT) suggest no difference in a variety of outcomes including ROSC, short-term survival, survival to hospital discharge, and survival with favorable neurologic outcomes with high dose compared to standard dose epinephrine, though there may be an improvement in ROSC [28-30]. However, one RCT found lower survival at 24 h with high dose epinephrine, with a trend to lower survival at discharge [31]. Based on this evidence, guidelines do not recommend use of high-dose epinephrine.

Several observational studies evaluating epinephrine in cardiac arrest suggest higher rates of ROSC but not necessarily improved survival at discharge or improved neurologic outcomes [32-37]. These observational data are also subject to significant selection and resuscitation time bias, as well as confounders. Several RCTs have also evaluated use of epinephrine, including the PACA trial of 601 patients with OHCA which evaluated epinephrine 1 mg every 3 min compared to placebo, finding improved ROSC but no difference or worse neurologic outcomes and no difference in survival to hospital discharge [38,39]. A meta-analysis including observational and RCT data evaluating prehospital use of epinephrine for OHCA found increased ROSC (OR 2.84, 95 % CI 2.28–3.54), but no difference in survival at 1 month and worse neurologic outcomes at discharge with epinephrine (OR 0.51, 95 % CI 0.31–0.84) [40]. The PARAMEDIC-2 trial included 8014 patients with OHCA randomized to epinephrine versus placebo [39]. Authors found improved 30-day survival with epinephrine versus placebo (3.2 % versus 2.4 %, unadjusted OR 1.39, 95 % CI 1.06–1.82). There was no difference in survival to discharge with a favorable neurologic outcome (2.2 % versus 1.9 %, unadjusted OR 1.18, 95 % CI 0.86–1.61). Severe neurologic impairment (defined as a modified Rankin scale of 4 or 5) occurred more frequently in the epinephrine group (31.0 % versus 17.8 %) [39].

Based on these data, epinephrine likely improves ROSC but not survival with favorable neurologic outcome. However, current guidelines continue to recommend epinephrine 1 mg every 3–5 min. Future studies should identify whether select patient groups are more likely to benefit from this, but based on current guidelines and data, administering epinephrine 1 mg every 3–5 min is reasonable [6-9].

2.3. Is the combination of vasopressin, steroids, and epinephrine beneficial?

Several other medications have been evaluated in cardiac arrest, including vasopressin and steroids. Previous reports have found higher endogenous vasopressin levels in patients who achieved ROSC compared to patients who died, though the clinical benefit of vasopressin in cardiac arrest remains controversial [41-44]. One RCT found no benefit with vasopressin compared to epinephrine in those with in-hospital cardiac arrest (IHCA) [45]. A second RCT evaluated patients with OHCA randomized to vasopressin 40 international units (IU) versus epinephrine 1 mg found no difference in hospital admission in those with VF

or pulseless electrical activity (PEA) [46]. However, in those with asystole, vasopressin was associated with higher rates of hospital admission when compared to epinephrine (29 % versus 20.3 %, $P = 0.02$) and hospital discharge (4.7 % versus 1.5 %, $P = 0.04$) [46]. Administration of epinephrine following two administrations of vasopressin was also associated with improved survival to admission (25.7 % versus 16.4 %, $P = 0.002$) and discharge (6.2 % versus 1.7 %, P = 0.002) [46]. However, a meta-analysis including 12 studies (6718 participants) evaluating IV vasopressin with or without epinephrine compared to epinephrine alone found no difference in ROSC (relative risk [RR] 1.11, 95 % CI 0.99–1.26), mid-term survival (defined as at hospital discharge, 28 days, 30 days, or 1 month) (RR 1.23, 95 % CI 0.90–1.66) and mid-term good neurological outcome (defined as cerebral performance scale 1–2 or modified Rankin scale 0–3) (RR 1.20, 95 % CI 0.77–1.87) [47]. The American Heart Association (AHA) guidelines state that vasopressin alone or in combination with epinephrine may be considered, but there is no advantage in using vasopressin as a substitute for epinephrine [7].

Global ischemia can result in systemic inflammation, and in the post-arrest period, low cortisol and reduced adrenocortical reserves are common [48-52]. Thus, steroids have been proposed as a treatment in cardiac arrest. While the 2020 AHA and 2021 European Resuscitation Council do not recommend the routine use of steroids in cardiac arrest [7,13], multiple RCTs have been published since those guidelines were released [53-56]. These RCTs suggest that steroids administered during and after cardiac arrest improve rates of ROSC, but the effects on survival or survival with favorable neurologic outcome are unclear [53-56]. A 2024 meta-analysis including 11 studies with 2273 patients with cardiac arrest found steroid administration during cardiac arrest was associated with an increased rate of ROSC (OR 2.05, 95 % CI 1.24–3.37) but not improvement in survival at discharge or survival with favorable neurologic outcomes [57].

Based on the purported benefits of vasopressin and steroids and the underlying pathophysiology in cardiac arrest, combining vasopressin, steroids, and epinephrine (VSE) has been evaluated as a potential combined intervention. A 2009 study enrolled 100 patients with IHCA in a double-blind, single center RCT [58]. Patients received vasopressin 20 IU and epinephrine 1 mg every CPR cycle compared with epinephrine plus saline placebo. At the first CPR cycle, patients in the intervention group also received methylprednisolone 40 mg versus placebo. If ROSC was obtained, patients received hydrocortisone 300 mg daily for up to 7 days with taper versus saline placebo. Authors found higher rates of ROSC (81 % versus 52 %, $P = 0.003$) and improved survival to discharge (19 % versus 4 %, $P = 0.02$) in the VSE group [58]. This study was followed by a larger RCT including 268 consecutive patients with cardiac arrest randomized to VSE versus placebo versus epinephrine [59]. Authors found higher likelihood of ROSC of 20 min or longer (OR 2.98, 95 % CI 1.39–6.40) and survival to discharge with a cerebral performance category score of 1 or 2 (OR 3.28, 95 % CI 1.17–9.20) [59]. A 2021 double-blinded multicenter RCT randomized 512 patients to VSE versus placebo with epinephrine, finding VSE was associated with higher ROSC (RR 1.30, 95 % CI 1.03–1.63, risk difference 9.6 %, 95 % CI 1.1 %–18.0 %), but there was no difference in 30-day survival or survival with a favorable neurologic outcome [53], and a post hoc analysis found no difference in favorable neurologic outcome at 6 months or 1 year [54]. A 2022 meta-analysis of 3 RCTs with 869 patients found improved ROSC with VSE compared to placebo (RR 1.32, 95 % CI 1.18–1.47), but there was no difference in survival to discharge [60]. A 2023 meta-analysis found similar results, with VSE associated with improved ROSC but no difference in mid-term survival or mid-term good neurologic outcome [47].

While evidence suggests improved ROSC with VSE or steroids with epinephrine, there is no improvement in survival or survival with favorable neurologic outcomes, and there is a dearth of evidence regarding VSE in OHCA or ED patients, as most studies have included patients with IHCA. Further randomized data are needed prior to the routine use of VSE or steroids with epinephrine in cardiac arrest in the ED.

2.4. What antiarrhythmics may have utility in cardiac arrest, and when should they be considered?

There are several antiarrhythmics available for cardiac arrest, including lidocaine, amiodarone, procainamide, and magnesium [12]. Antiarrhythmics are most commonly used in those with pulseless ventricular tachycardia (pVT)/ventricular fibrillation (VF) that is refractory to defibrillation [6-9,12].

Lidocaine is a Class Ib antiarrhythmic that may be used in those with refractory pVT/VF. Dosing in cardiac arrest includes 100 mg, followed by 50 mg after the fifth shock. Data from observational studies evaluating OHCA and IHCA suggest lidocaine may improve survival at 1 year and survival with favorable neurologic outcome [61,62]. However, RCT data suggest that lidocaine is not superior to amiodarone or placebo for survival to discharge and survival with favorable neurologic outcome [63-66]. A Bayesian reanalysis of the ALPS RCT found a modest treatment benefit with lidocaine for improved survival and neurologic outcome [67].

Amiodarone is an antiarrhythmic with Class I, II, and III activity. Dosing in cardiac arrest includes a 300 mg IV bolus, followed by 150 mg IV bolus as needed. A 1999 study evaluating amiodarone versus placebo in OHCA with refractory rhythms found a higher rate of survival to admission with amiodarone compared to placebo (44 % versus 34 %, OR 1.6, 95 % CI 1.1–2.4) [68]. A 2002 study evaluating 347 patients found amiodarone compared to lidocaine was associated with higher survival to admission (22.8 % versus 12 %, OR 2.17, 95 % CI 1.21–3.83) [65]. A 2016 double blind, placebo-controlled RCT with 3026 OHCA patients found amiodarone and lidocaine did not improve survival or favorable neurologic outcome compared to placebo [63]. The 2017 ALPS trial of 3026 patients with initial pVT/VF and 1063 with initial nonshockable-turned-shockable rhythms found amiodarone and lidocaine were not associated with improved survival to discharge compared to placebo, though amiodarone was superior in patients with witnessed OHCA on subgroup analysis (absolute risk difference 21.9 %, 95 % CI 5.8–38.0) [64]. Several secondary analyses of the ALPS trial have been conducted. A subgroup analysis evaluating antiarrhythmic medications based on vascular access (IV versus IO) found that when adjusted for confounders, amiodarone IV was associated with improved survival to admission, survival to discharge, and survival with hospital discharge. However, this improvement was less pronounced in patients receiving lidocaine IV, with no improvement in those receiving amiodarone or lidocaine via the IO route [69]. A third subgroup analysis found that the likelihood of ROSC was greatest with antiarrhythmic administration within the first 10 min from the time of the 911 call (OR 0.92, 95 % CI 0.90–0.94 per minute increase) [70]. A Bayesian reanalysis of the ALPS trial found that amiodarone was highly likely to improve survival to hospital discharge and neurologic outcome when compared to placebo [67].

Procainamide is a Class Ia antiarrhythmic that was previously a commonly used agent for cardiac arrest with refractory pVT/VF. However, procainamide has a slower infusion rate and may have adverse reactions (e.g., QRS/QT prolongation, VT, VF, complete atrioventricular block, torsades de pointes) [12]. Dosing in cardiac arrest is 20–50 mg/min until pVT/VF resolves or a maximum of 17 mg/kg is reached. The Procainamide versus Amiodarone for the Acute Treatment of Tolerated Wide QRS Tachycardia (PROCAMIO) trial evaluated stable patients with ventricular tachycardia randomized to procainamide (10 mg/kg/20 min) or amiodarone (5 mg/kg/20 min) and found procainamide was associated with less major cardiac adverse events (OR 0.1, 95 % CI 0.03–0.6) and greater likelihood of terminating the tachycardia within 40 min (OR 0.49, 95 % CI 0.15–1.61) [71]. There have been no RCTs evaluating procainamide for shock-refractory OCHA. However, a 2010 retrospective study with 665 patients evaluating procainamide for refractory

pVT/VF found no improvement in survival to discharge [72]. A second 2022 retrospective study evaluating procainamide, amiodarone, and lidocaine found procainamide had higher prehospital ROSC but similar ED ROSC and survival [73].

Magnesium is an electrolyte that assists in regulating the flow of sodium, potassium, and calcium across cellular membranes, and it also functions as a cofactor for several metabolic reactions with adenosine triphosphate [12]. Several RCTs and observational studies have evaluated magnesium compared with placebo for patients with OHCA and IHCA in nonshockable and shockable rhythms [74-81]. However, these studies have not found improved outcomes including ROSC or survival to discharge [74-81]. Magnesium is a treatment for torsades de pointes (pVT with long QT interval) based on limited data [82,83]. However, magnesium primarily serves to prevent reinitiation of torsades, rather than converting the arrhythmia [12]. Dosing for magnesium sulfate is 2–4 g IV, followed by infusion of 1 g/h targeting serum magnesium 3.5–5 mg/dL [12].

Several meta-analyses evaluating antiarrhythmics in cardiac arrest have been conducted. A 2018 systematic review with 14 RCTs and 17 observational studies evaluating amiodarone, procainamide, lidocaine, magnesium, and bretylium found no improvement in survival to discharge, survival with favorable neurologic outcomes, and long-term survival [66]. A separate systematic review of 30 studies (39,914 participants) with 8 antiarrhythmics found no improvement in ROSC, survival to admission, survival to discharge, or survival with favorable neurological outcomes [84].

Based on the current evidence, antiarrhythmics likely do not improve short-term or long-term survival or survival with favorable neurologic outcome. However, guidelines continue to incorporate antiarrhythmics in cardiac arrest, and they can be considered for patients with refractory pVT/VF.

2.5. What is the utility of calcium administration in cardiac arrest?

Calcium has been proposed as a treatment in cardiac arrest due to its inotropic and vasopressor effects, and it may reduce the proarrhythmic effects of hyperkalemia [12]. One study found an increase in calcium administration in patients with IHCA from 2001 to 2016 [85]. However, several studies have demonstrated no benefit and potential harm with routine administration of calcium chloride in patients with cardiac arrest. The Calcium for Out-of-Hospital Cardiac Arrest (COCA) trial was a double-blind, placebo-controlled RCT randomizing patients with OHCA [86]. Patients were randomized to calcium chloride (up to 2 doses of calcium chloride 5 mmol given IV or IO) or placebo, administered after the first dose of epinephrine. Following a preplanned interim analysis with 383 patients, an independent safety committee stopped the study early due to harm in the calcium group. Authors found no difference with calcium versus placebo in sustained ROSC (19 % versus 27 %, RR 0.72, 95 % 0.49–1.03), 30-day survival (5.2 % versus 9.1 %, RR 0.57, 95 % 0.27–1.18), or 30-day survival with favorable neurologic outcome (3.6 % versus 7.6 %, RR 0.48, 95 % 0.20–1.12) [86]. However, there was a trend towards worse outcomes in patients who received calcium. A long-term analysis of patients from the COCA trial analyzed 391 patients and found no difference in survival (4.7 % versus 9.1 %, RR 0.51, 95 % CI 0.24–1.09), but a reduced likelihood of favorable neurologic outcome at one year with calcium (3.6 % versus 8.6 %, RR 0.42, 95 % CI 0.18–0.97) [87]. A preplanned subgroup analysis of patients with PEA potentially associated with hyperkalemia and ischemia found a trend towards lower ROSC with calcium administration (20 % versus 39 %, RR 0.51, 95 % CI 0.26–1.00) [88]. Finally, a systematic review of 4 studies with 554 adult patients with OHCA, 8 observation studies with 2731 adult patients with OHCA, and 3 observational studies with 17,449 pediatric IHCA patients found no benefit with calcium administration [89]. Based on the available evidence, calcium should not be routinely administered in those with cardiac arrest, unless hyperkalemia is the suspected cause.

2.6. What is the utility of sodium bicarbonate administration in cardiac arrest?

Sodium bicarbonate can increase circulating blood volume and is thought to improve the acidosis that may develop in cardiac arrest with reduced circulation [12]. The use of sodium bicarbonate in IHCA has increased, with close to 50 % of patients with IHCA receiving sodium bicarbonate in 2016 [85]. However, there are no high-quality data supporting the routine use of sodium bicarbonate in patients with cardiac arrest. A 2021 meta-analysis including 4 RCTs and 10 observational trials with a total of 28,412 patients in OHCA found no improvement in ROSC or survival to hospital discharge with sodium bicarbonate administration in cardiac arrest, and it was associated with lower rates of sustained ROSC (OR 0.27, 95 % CI 0.07–0.98) and favorable neurologic outcomes at discharge (OR 0.12, 95 % CI 0.09–0.15) [90]. A 2023 RCT of 6 studies (3 RCTs and 3 propensity score matched cohort studies) with 21,402 patients found no difference in short-term or long-term survival with sodium bicarbonate [91]. Current guidelines also recommend against the routine administration of sodium bicarbonate in cardiac arrest [6-9,92]. Thus, based on the current literature, sodium bicarbonate should not routinely administered in cardiac arrest. However, there are several conditions in which sodium bicarbonate may be used, including hyperkalemia and sodium channel blocker toxicity (e.g., tricyclic antidepressant overdose).

2.7. Are beta-blockers useful in shock-resistant pVT/VF?

Beta-blockers have been evaluated for treatment of refractory pVT/VF, as beta-blockade may counteract the deleterious effects of excess sympathetic and beta-adrenergic stimulation that occurs with repeated doses of epinephrine [93-96]. Beta-blockers may include esmolol 300–500 μg/kg IV bolus followed by 50–200 μg/kg/min infusion, metoprolol 2.5–5 mg IV every 2–5 min to a maximum of 15 mg, or propranolol 0.15 mg/kg over 10 min followed by 3–5 mg every 6 h [12]. A meta-analysis of 3 studies with 115 patients found increased temporary ROSC (OR 14.46, 95 % CI 3.63–57.57), sustained ROSC (OR 5.76, 95 % CI 1.79–18.52), survival-to-admission (OR 5.76, 95 % CI 1.79–18.52), survival-to-discharge (OR 7.92, 95 % CI 1.85–33.89), and survival with favorable neurologic outcome (OR 4.42, 95 % CI 1.05–18.56) [93]. However, the risk of bias was moderate to severe, and the overall certainty of evidence was low. A trial sequential analysis of these studies in the meta-analysis recommended that further studies are required prior to the routine use of beta blockers in refractory pVT/VF [94]. Based on the available evidence, beta-blockers may be considered in those with shock-resistant pVT/VF, though further study is required.

3. Conclusions

Medications are a component of cardiac arrest management. Administration via the IV route is preferred. If IV access is not feasible or unsuccessful, then the IO route should be utilized. Epinephrine, VSE, and antiarrhythmics have demonstrated improved rates of ROSC but not survival to discharge or survival with a favorable neurologic outcome. Epinephrine is recommended by ACLS guidelines. Antiarrhythmics such as amiodarone may be used in those with refractory pVT/VF. Calcium and sodium bicarbonate should not be administered routinely in cardiac arrest. Beta-blockers may be considered in shock-resistant pVT/VF.

CRediT authorship contribution statement

Brit Long: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Formal analysis, Conceptualization. **Michael Gottlieb:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Formal analysis, Conceptualization.

Declaration of competing interest

None.
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